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THE COURSE OF TRAUMATIC SHOCK IN DOGS SUSTAINING PROLONGED HYPODYNAMIA

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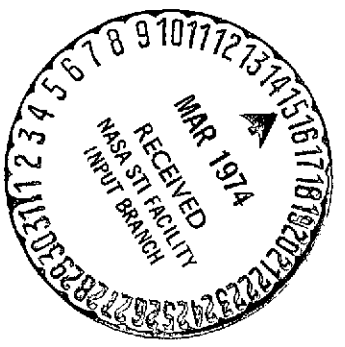

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16. Abstract

Following a 2-week hypodynamia, traumatic shock developed in dogs after infliction of a somewhat greater trauma than in the control, but, following a 28-day hypodynamia, much less trauma was required for development of shock. Regardless of duration of hypodynamia, the survival period of experimental animals was much less than that of the controls.

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THE COURSE OF TRAUMATIC SHOCK IN DOGS SUSTAINING PROLONGED HYPODYNAMIA

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It is known that prolonged hypodynamia significantly changes /83* the reactivity of the body and reduces its resistance to a number of pathogenic actions [1-2]. However, we have not succeeded in encountering data in available literature, disclosing its importance for traumatic shock to any extent.

Method

The work was carried out on 31 female dogs, weighing 13-24 kg. Hypodynamia was induced by restraining the dogs in special stalls, sharply restricting their motor activity. Ten dogs (series I) were under hypodynamia conditions for 14 days and eight dogs (series II), 28 days. After removing the animals from the stalls, they were secured on a table and subjected to shock-inducing trauma, by means of crushing the soft tissues of the thigh until disappearance of a defensive reaction, development of a general serious condition and reduction of arterial pressure to 60-65 mm. The same shock was induced in 13 intact dogs (series III, control). The arterial pressure from the central end of the right femoral artery and respiration, by means of a Marey capsule and cuffs slipped over the chest of the animal, were recorded on kymograph tape. Besides, the sinocarotid pressor reflexes, in response to /84 pinching the common carotid artery for 5 and 10 sec, were determined. After inflicting the trauma, the animals were observed for a period of 6 hours and their survival time in shock was noted.

* Numbers in the margin indicate pagination in the foreign text.

Results and Discussion

In the initial state, the average arterial pressure level in dogs sustaining hypodynamia was somewhat higher (series I 166 ± 5.4; $P < 0.05$; series II 153 ± 4.2 mm), than in the controls (148 ± 4.5 mm). The respiration rate in dogs after 28 days of hypodynamia (28 ± 4) was considerably less ($P < 0.05$) than in the remaining series of tests (control 54 ± 9; series I 66 ± 14).

After infliction of the trauma, following brief agitation in the animals, a general serious condition developed, the sino-carotid pressor reflexes disappeared or underwent phase changes, arterial pressure decreased and marked tachycardia and curtailment of respiration were observed. In the majority of cases, immediately after the trauma third order waves appeared on the arterial pressure record curve, which became deeper in proportion to development of the shock and disappeared 30-40 min before death of the animals.

Under our conditions, for reproduction of the shock in animals after 28 days of hypodynamia, considerably less trauma had to be applied ($P < 0.01$) than in all the remaining ones. The magnitude of the trauma inflicted on dogs of test series I and III did not differ significantly.

The magnitude of the shock-inducing trauma in series I was 175 ± 24, 87 ± 9 in series II and 140 ± 16 blows in III.

In all dogs sustaining hypodynamia, no distinct tendency towards increase in arterial pressure was noted after the trauma, a more general depression progressed and, after 60-90 min, as a rule, they died. In distinction from this, in the majority of control animals, quite a prolonged period of relative compensation was observed, and their survival times in the state of shock

(197 ± 30 min) significantly ($P < 0.01$) exceeded that in dogs sustaining hypodynamia (series I 68 ± 18, series II 86 ± 15 min). Two dogs of the control series of tests independently came out of shock, which did not occur in tests with hypodynamia.

Thus, prolonged hypodynamia changes the reactivity of the body considerably, with respect to extreme conditions of mechanical shock-inducing trauma.

The higher initial arterial pressure level in the controls after 14 days of hypodynamia is evidence of either stronger stress mechanisms of the neuroendocrine regulation of the cardiovascular system or of reduction in the cholinergic effects on the latter. The possibility is not excluded that both of these mechanisms occur.

In the first 2 weeks of hypodynamia, symptoms of systemic emotional stress were noted. This possibly explains the necessity for inflicting a somewhat larger number of blows to the animals after 14 days of hypodynamia than in the controls, since systems responding to neuroendocrine regulation are already prepared non-specificially to some extent for the action of extreme stimuli. However, regardless of the length of hypodynamia, the survival time of the animals after trauma is considerably less than in the control. Thus, after restriction of mobility, the capability of the cardiovascular system for support of arterial pressure after trauma, even if minimally low, consistent with the level of life, is restricted, and generalized vascular collapse sets in quickly.

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